ABSTRACT

The present inventors revealed by using fasted and refed mice, that AdipoR1/R2 is a regulator of metabolic sensitivity to nutritional conditions and insulin. They showed that mRNA level of AdipoR1/R2 increased by STZ treatment, and that this increase was restored by insulin. The present inventors confirmed *in vitro* that insulin reduces AdipoR1/R2 mRNAs in myocytes and such. It was also confirmed that in insulin-resistant models, the AdipoR1/R2 expression was downregulated, and that AMP kinase activation by adiponectin was decreased. The present inventors discovered by using insulin signaling pathway inhibitors, that the downregulation of adiponectin receptors by insulin was mediated by the PI3-kinase/Foxo1 pathway.